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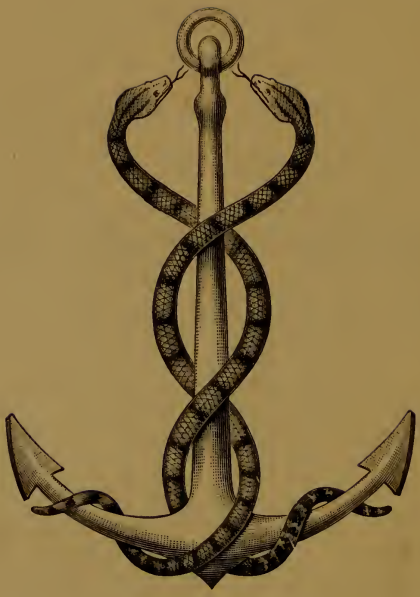
THE MURMURS OF MITRAL DISEASE.



NUNQUAM ALIUD NATURA, ALIUD SAPIENTIA DICIT.

With my compliments

THE MURMURS

OF

E. M. Brockbank

MITRAL DISEASE

BY

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PREFACE

THE most important portion of this small work has already appeared in the pages of the *Edinburgh Medical Journal*, in the March and April numbers of this year, as a paper entitled "The Causation and Rhythm of the Crescendo Murmur of Mitral Stenosis."

To this paper I have added a brief chapter on some of the literature of the subject, and another containing a short criticism of Sir Wm. Gairdner's classical case of tricuspid obstruction. I am greatly indebted to Sir Wm. Gairdner for permission to reproduce one of the plates by which he illustrated the paper referred to in Chapter V.

I venture to suggest, in justification of this small work, that it is a study of the much debated question of the crescendo murmur from the standpoint of a new theory of origin of this murmur, namely, that promulgated by myself in the *Manchester Medical Chronicle* for June 1897. And the fact that, by such new arguments, the early systolic rhythm theory of the murmur receives additional support to that afforded by previous observers looking at the question from different points of view, is, to my mind, further evidence of the justness of such a theory.

My thanks are due to the Honorary Physicians of the Manchester Royal Infirmary for their kindness in allowing me to examine the cases under their charge in the wards of the hospital.

E. M. B.

ST. PETER'S SQUARE, MANCHESTER,
May 1899.

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THE MURMURS OF MITRAL DISEASE.

CHAPTER I.

SOME PREVIOUS CONTRIBUTIONS TO THE LITERATURE OF THE PRESYSTOLIC OR CRESCENDO MURMUR OF MITRAL STENOSIS.

The literature of the presystolic theory question is so well known that I need only refer here to a few of the important contributions to it.

Fauvel,[1] in 1843, was the first to apply the term presystolic to a peculiar cardiac bruit localised at the apex beat of the heart, and which he thought to be most probably a stethoscopic sign of contraction of the mitral orifice.

Walshe,[2] in 1851, described a post-diastolic or presystolic murmur of maximum force immediately above and about the left apex, and conducted on the same principle (though less extensively) as the systolic murmur of the same seat, as being an indication of obstructive narrowing of the mitral orifice, or of simple roughness of the auricular surface of the mitral valve, or of both these conditions.

But it was not until 1861, when Gairdner published in the *Edinburgh Medical Journal*, in a paper on "A Short Account of Cardiac Murmurs," and in 1862, in his work on "Clinical Medicine," a clear description of the auriculo-systolic murmur, as he called it, that the more generally accepted theory of the causation and rhythm of the murmur began to be accepted by English physicians.

Gairdner's description of the murmur is as follows :[3]—

"The murmur precedes and runs up to the first sound

[1] *Arch. gén. de méd.*, Paris, Sér. 4, tome i. p. 1.

[2] "Diseases of the Heart," 1856, p. 226.

[3] *Op. cit.*, p. 574.

ending at the moment of this sound and of the apex beat. In this case the murmur is simultaneous with the contraction of the auricles, and I call it auricular-systolic. The interpretation of such a murmur depends on the consideration, that it occurs *only when blood is being expelled from an auricle*, and when the ventricle is passive. With very rare exceptions, therefore, such murmurs depend upon contraction of the auriculo-ventricular orifices, and consequent interruption to the flow of blood out of the auricle during its contraction. The auricular-systolic murmur may merely precede the first sound, *i.e.*, it may follow the pause of the heart's action; or it may appear to be prolonged out of, or even quite through, the period of rest, being in this last case necessarily associated with a degree of the ventricular-diastolic murmur, presently to be described. Its essential character, however, is preserved in every case, as above defined."

The nomenclature adopted by Gairdner has a misleading tendency, and in 1893 [1] he explained that the name auricular-systolic was applied to the latter, or prolonged variety, of the murmur, not "because it was *produced by* the auricle in systole, but simply because the most characteristic element of the murmur, the so-called presystolic rhythm, was, in fact, *coincident in time* with the auricular systole."

In 1864, Ormerod [2] expressed his doubts as to the truth of Gairdner's views, and thought that there was no insuperable objection to another explanation of the murmur in question. Its seat at the mitral or tricuspid orifice is undoubted, but Ormerod thought that the murmur might arise from "imperfection of the auriculo-ventricular valve, allowing regurgitation during the contraction of the ventricle."

Fagge [3] published in 1871 an important contribution to the discussion, giving a full account of the previous papers on the subject, and supporting the presystolic theory of the rhythm of the bruit. In his experience "the murmur is occasionally so short and sudden that it resembles rather a tone, and is in itself hardly to be distinguished from the natural first sound of the heart."

The early systolic theory received further strong support from Barclay [4] in 1872, and four years later from Charlewood Turner.[5]

[1] *Edin. Hosp. Rep.*, vol. i. p. 230, note 1.
[2] *Med. Times and Gaz.*, London, 1864, vol. ii. p. 154.
[3] *Guy's Hosp. Rep.*, London, Ser. 3, vol. xvi. p. 247.
[4] *Lancet*, London, 1872, vol. i. p. 283.
[5] *London Hosp. Rep.*, 1876, New Series, vol. vii. p. 127.

More recently (1887) a very important contribution to the discussion was made by Howship Dickinson[1] in a paper entitled "On the Presystolic Murmur, falsely so called." In this paper, Ormerod's and Barclay's views were supported by very powerful arguments, derived from much clinical experience. It is impossible to do justice to Dickinson's views in a short abstract of his paper, but I may note that one of his strongest reasons for believing that the "presystolic" is really a ventricular-systolic murmur is the frequently repeated observation, that "if a finger be pressed between the ribs, particularly in a thin person, and when the heart is not too rapid, and if, at the same time, the double stethoscope be adjusted to the murmur, the beginning of the murmur will be found synchronous with the beginning of the impulse."

Dickinson looks upon the "short sound which immediately follows the special murmur—up to which the latter runs—as marking the conclusion of ventricular systole."

This point as to the exact place in the cardiac rhythm at which the short, abrupt first heart sound occurs, is very important; and as I discuss it fully at a later page, I will only say here, that I believe Dickinson to be in error in placing it at the *conclusion* of ventricular systole.

Dickinson's original papers, "On the Presystolic Murmur, falsely so called," appeared in the *Lancet*, London, in 1887[2] and 1889,[3] and excited considerable correspondence in favour of and against his theories, in which all the authorities on the subject joined; to these pages I beg to refer any reader who wishes for wider references to the literature of this subject than I am giving here.

Of recent years a good deal of attention has been paid to the action of the auricle in cases of mitral stenosis. It is stated that in health, instead of ceasing its contraction before that of ventricular systole begins, as used to be thought the case, the auricle may continue to remain contracted after the commencement of the contraction of the ventricular muscle, "both auricle and ventricle continuing to contract simultaneously until the moment when the sigmoid valves are opened and blood begins to be expelled from the ventricle into the aorta. Potain considers that the auricle is in action from the beginning of its systole until the precise moment of closure of the auriculo-ventricular valves—that it is this

[1] "Occasional Papers on Medical Subjects," 1896, p. 225.
[2] Vol. ii. p. 650. [3] Vol. ii. p. 799.

muscular contraction of the auricle which ordinarily causes the propulsion of the heart's apex against the wall of the chest, and that thus it plays a notable part in the production of the impulse which is felt by the hand applied over the situation of the apex beat. In stenosis of the mitral aperture, this lifting of the apex by the force of the contracting auricle may be greatly exaggerated (Sansom).[1] Samways believes that the abnormally powerful contraction of the left auricle prevents regurgitation in compensated mitral stenosis, and further, that the auricle may even be strong enough to force blood into the ventricle, after contraction of the latter has begun."[2] These same observers believe that this prolonged auricular contraction prevents regurgitation of blood during early ventricular systole, and therefore puts the possibility of the crescendo murmur being systolic in rhythm out of the question. Woods,[3] criticising Samways' view that the auricle can contend successfully with the ventricle, when their contractions overlap, and send blood into the ventricle at that time, forms the opinion, from his own observations with similar experiments to those from which Samways drew his conclusions, that such a theory is "absurd." He says that there are no valves between the auricle and the lungs, and therefore, raising the intra-auricular pressure during contraction of the ventricle could only result in the blood following the path of least resistance, namely, back into the pulmonary veins. Woods also gives mathematical reasons for believing Samways' theory of the greater strength of the auricular compared with that of ventricular contraction to be "absurd."

I fail to see how the contracting auricle could possibly prevent the regurgitation of the very small stream, and very small total amount, of blood which alone is necessary to produce many audible crescendo murmurs. And even were this contention unquestionable when the auricular wall is normal or hypertrophied, it is impossible, to my mind, to understand how the thin atrophied muscle of the auricular wall, so often met with in mitral stenosis, and which, in Sansom's experience, may be represented by only a few muscular fibrillæ scattered through a shell of fibrous tissue, can contract so forcibly over the mitral orifice as to prevent the regurgitation of blood through the stenosed valve, under the great force of ventricular systole.

[1] Allbutt, "System of Medicine," vol. v. p. 1014.
[2] *Ibid.*, vol. v. p. 1015.
[3] *Brit. Med. Journ.*, London, 1897, vol. i. p. 627.

A theory which explains the prevention of regurgitation at the onset of ventricular systole in mitral stenosis must hold good, not only when the auricle is hypertrophied or even of normal thickness, but also when the auricular wall, as so often is the case, is atrophied or abnormally thin; and the above theory, to my mind, entirely fails in this.

But although the question of the rhythm of the so-called presystolic murmur has been so much discussed in the past fifty years, as far as I can find, no explanation of the causation of the very important characteristic of ascending pitch, which the murmur always possesses, was offered until I published a paper on this subject in June 1897.[1]

The method of causation of the murmur in question is, in the present writer's opinion, all-important, for once the means by which the murmur obtains its peculiar characteristics be recognised, the simpler becomes the attempt to define the true rhythm of the murmur. This theory is dealt with fully in the next chapter.

Cowan, in 1898,[2] propounded a theory which agrees with my contention, that the ascending pitch characteristic of the crescendo murmur is due to blood being forced through the mitral valve whilst its orifice is being rapidly diminished in lumen, and also with the view held by others as well as by myself, that the terminal abrupt sound is caused by the impact of the hard roughened edges of the diseased mitral valve.

He believes, however, that the crescendo murmur of mitral stenosis is caused during ventricular systole by blood flowing from the auricle through the mitral valve "into the ventricle whilst changes in the tension of the valves and alterations in the lumen of the orifice are taking place, the result of contraction of the ventricle as a whole, and of the musculi papillares in particular." Cowan is of the opinion that "at the commencement of ventricular systole the pressure of the blood in the ventricle will be slight compared with that in the auricle, and until the ventricular pressure rises above the auricular pressure, as the result of the ventricular systole, the valves will not be closed, and blood will flow into the ventricle from the auricle."

The objections to such a theory are practically the same as those offered to Samways' views on the previous page.

[1] *Med. Chron.*, Manchester, June 1897.
[2] *Glasgow Med. Journ.*, March 1898.

CHAPTER II.

THE CAUSATION AND RHYTHM OF THE CRESCENDO MURMUR OF MITRAL STENOSIS.

DEFINITION.—By the crescendo murmur of mitral stenosis, I mean that abnormal heart sound generally called "presystolic," and less often "auricular systolic," which is practically diagnostic of obstructive narrowing of the auriculo-ventricular valve orifices, especially on the left side of the heart. Though the true crescendo murmur is diagnostic of stenosis of the mitral and tricuspid valves, several observers have described a murmur which they call presystolic, as occurring in other cardiac lesions, and even in dilatation of these same valves. I shall deal with this latter murmur in a future paper, and in the meantime will confine my remarks to the true crescendo murmur of mitral or tricuspid stenosis, and especially of the former affection.

NATURE OF THE MURMUR. — The characteristics of the crescendo murmur are known to everyone, but there are one or two points to which I wish to direct special attention. The murmur is short and vigorous, and evidently produced by some strong force. It immediately precedes, and terminates abruptly with, the closure of the mitral valve, an event which is generally marked by an accentuated first sound of the heart, and invariably by the true cardiac impulse. The essential and most peculiar characteristic of the murmur is that it rises progressively from low to high pitch, like an ascending scale of notes, as it rushes rapidly to its abrupt termination. It is also generally of a true crescendo character—that is, it gathers in power of tone coincidently with its progressive rise in pitch. By the abrupt termination of the murmur at its stage of most complete development, the two important features of rising pitch and increasing force are emphasised.

There are, broadly speaking, two types of crescendo murmur, both of which terminate abruptly with the first sound of the heart,

one rough and bur-r-ing or spluttering, and the other smoother and more whiffy in character, the latter type being often overlooked by observers. Some murmurs are accompanied by a tactile thrill, which can be palpated at the apex beat of the heart. The rhythm of this thrill is identical with that of the murmur which it accompanies.

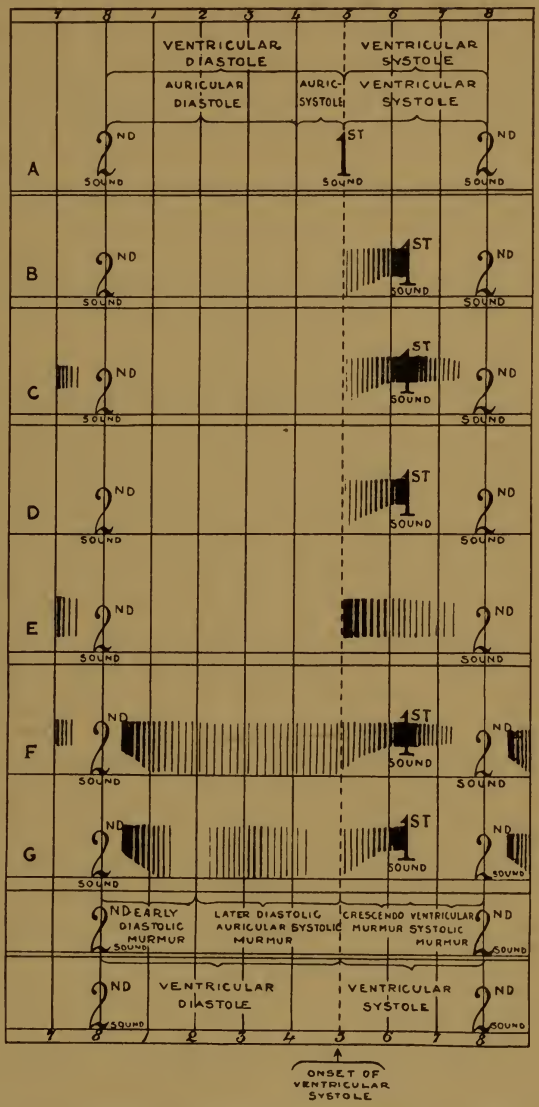

FIG. 1.—Diagram showing the rhythm and the relative duration of the events of a normal cardiac cycle, and of the murmurs of mitral stenosis in a heart beating 75 times per minute. Each numbered space represents ·1 second; each cardiac cycle occupies ·8 second.

Though of variable duration, the true crescendo murmur of mitral stenosis occupies at most ~~not more~~ than half the interval between the second and first heart sounds (Fig. 1, B, C, D, F, G). But it may appear to be much longer than this, for not infrequently it is apparently continuous with an ordinary diastolic mitral bruit of quite different type, namely, uniform or decrescendo. In such cases murmurs may be heard to occupy the whole

of the interval between the second and first heart sounds, either as one long continuous sound or with a slight break between the component bruits (Fig. 1, F, G). Some eminent authorities call this whole composite murmur crescendo, presystolic, or auricular systolic, but such a nomenclature is confusing, as they also apply the same terms to the true crescendo murmur of ascending pitch when it occurs, as it does in the majority of cases, quite separately in the latter portion of the interval between the second and first heart sounds, and immediately before the latter sound. But, be this as it may, I am only concerned with the undoubted crescendo murmur of ascending pitch in this paper, whether it occurs alone or as part of a composite murmur, and I shall confine my remarks almost entirely to this peculiar murmur.

In the accompanying diagram (Fig. 1) of the rhythm of the murmurs of mitral stenosis, I have departed slightly from the arrangement of lines of various length, thickness, and closeness together by which Gairdner[1] first represented schematically the varying intensity, rhythm, and duration of these murmurs.

The varying length of the lines in my diagrams represents the comparative *areas* of the stenosed orifice during the production at it of the different types of murmur; the thickness of the lines and their closeness together indicate the *force* of the murmur; and, finally, the elevation of the lines above the base line represents the rise or fall in *pitch* which the crescendo and mitral diastolic murmurs undergo. Thus the diagram of a crescendo murmur (Fig. 1, B, D), interpreted according to the above explanation, signifies that this bruit begins at the very onset of ventricular systole (the line 5), becomes rapidly more and more vigorous, and of higher and higher pitch, as it rushes to its abrupt termination with the accentuated first sound of the heart, and that the orifice at which it is created is open to its widest extent at the beginning of the murmur, and closed, or almost completely closed, at the appearance of the accentuated terminal sound.

Exactly the opposite sequence of events is met with in the development of the early diastolic decrescendo murmur, as indicated by the diagram (Fig. 1, F, G), whilst the later diastolic murmur, part of which is produced (Fig. 1, F, G) in the period of auricular systole, is created at an orifice which remains as widely open as possible during the course of the murmur, and is the result of the action of forces which vary in intensity, and

[1] "Clinical Medicine," 1862, p. 574.

which generally die away more or less before the onset of ventricular systole.

The ordinary systolic murmur of regurgitation in mitral stenosis (Fig. 1, E) is of a lower pitch, and produced at an orifice of greater area of leakage than is the case when the systolic murmur continues the abruptly-terminating crescendo murmur (Fig. 1, C, F).

CAUSATION AND RHYTHM.—Two theories are advanced in explanation of the rhythm, and, to a less extent, of the causation of the crescendo murmur.

1. The *presystolic* or original and orthodox view is that "the murmur is due to the extrusion of blood through the stenosed aperture, under pressure by the auricle, aided by the aspirating force of the ventricle, during the period of diastole of the heart; in most cases it suddenly ceases at the moment of the production of the first sound of the heart; it manifests a heightened intensity at its close, due to the contributory impulsive force of the auricular systole; the first sound which terminates it is usually very short and abrupt, and is due chiefly to the sudden tension of the tricuspid valve and of the walls of the right ventricle."[1]

2. The *early systolic* or other theory is, that the murmur arises from blood escaping through the mitral valve, before it is closed by ventricular systole, and that it is early ventricular systolic in rhythm.

This latter theory, though looked upon as in a way heretical, is gaining more and more supporters; and, though brought up in orthodoxy myself, I have been obliged to join the smaller band of unbelievers. Some of my reasons for doing so were given in a previous paper,[2] and the object of the present communication is to mention further arguments which appear to me to bring more support to this early systolic rhythm theory of the crescendo murmur.

It will be advisable for me to sketch briefly the main points of my former paper. In it I started with an entirely new explanation of the causation of the ascending-pitch characteristic, which is so typical of the crescendo murmur of mitral stenosis. The theory I then stated is, that *the crescendo murmur of mitral stenosis is produced by blood regurgitating through the stiff, rigid orifice of the narrowed valve, whilst this orifice, which resists closure,*

[1] Sansom, "Diseases of the Heart and Thoracic Aorta," 1892, p. 378.

[2] *Med. Chron.*, Manchester, June 1897, p. 161.

is being rapidly diminished in area and finally obliterated by the action of a strong force, which on its part increases progressively in strength with the duration of the bruit.

The main point in this theory is, that the ascending pitch of the murmur is imparted to it by the blood rushing through an orifice which is being gradually diminished in area of lumen, and to illustrate this contention I described a simple experiment with a piece of drainage tubing about $1\frac{1}{2}$ in. long and $\frac{1}{3}$ in. to $\frac{1}{2}$ in. in diameter of lumen. If the observer will place such a piece of tubing between his teeth, and, with his lips closely applied to it, blow through it, he will produce a sound or bruit; and if, whilst producing this bruit, he brings his teeth together and obliterates the lumen of the tube (Fig. 2), the character of the bruit will change from low to higher pitch, until it is finally cut off when at its stage of highest pitch, by the obliteration of the lumen of the tube. The movement of the teeth must not be too rapid, but occupy, to begin with, about 1 to $1\frac{1}{2}$ seconds, in which case the changes in pitch are very noticeable. At the same time, by varying the rapidity by which the tube is obliterated, the character of the resulting bruit varies within limits similar to those of a natural crescendo murmur.

If the above series of changes be carried out rapidly, the murmur so produced terminates with a thud, caused by the collision of the walls of the tube; and this point I used as experimental evidence in favour of the theory, that the accentuated first sound of the heart, which terminates a crescendo murmur, is due to the impact of the walls of the thickened rigid orifice of the valve.

The intensity of the artificial sounds varies with the force used to produce them, and with the abruptness with which the teeth obliterate the lumen of the tube; and the greater the diameter of the tube, the lower will be the pitch of the note produced by blowing through it, and *vice versâ*. If in manufacturing the crescendo murmur, in the way described above, the teeth be moved together more carefully, so that, whilst the lumen of the tube is practically obliterated, its walls are not brought into violent collision, the crescendo murmur will still be heard, but without a terminal thud.

Finally, if the teeth be brought together so as to cut off the greater portion of the lumen of the tube, but to leave a slight area unobliterated (Fig. 2, E, F), the crescendo murmur and a modified terminal thud will still be produced, but running on

from the thud will be heard a high-pitched, uniform murmur, which is caused by the escape of air through the "leak" in the tube. This last bruit is an experimental imitation of the systolic murmur which not uncommonly runs on continuously from a crescendo murmur in mitral stenosis. This simple experiment, to my mind, explains away very clearly what is to many observers a stumbling-block to the acceptance of the systolic rhythm theory of the crescendo murmur, namely, that it is impossible for even an abnormal first sound of the heart to appear in what is apparently the middle of ventricular systole, and to separate two murmurs, each of which is also produced during ventricular systole.

With the above simple experiment in mind, such an event may easily be explained, and I have shown diagrammatically in Fig. 1, C, F, the rhythm of such a compound bruit in mitral

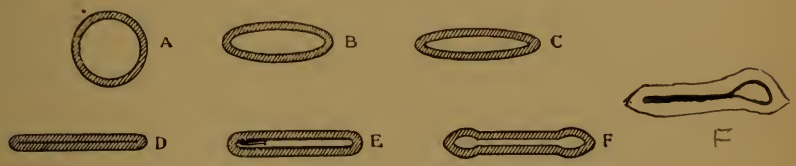

FIG. 2.—A, B, C, D, Progressive stages in the obliteration of the lumen of a tube in which is being produced an imitation crescendo murmur; E, F, condition of the lumen of the tube when a uniform murmur continues the crescendo murmur.

stenosis. The increased vigour which also characterises a crescendo murmur is imitated by increasing the force with which the air is blown through the tube in the production of the imitation bruit. All the above experiments with air bruits can be carried out with the lips, if they be pursed up as if for producing a whistle of low pitch, a method which is frequently adopted by teachers for giving students a good idea of what cardiac murmurs are like.

Having in this way explained the causation of the most important characteristics of the murmur, I went on to consider what must be the part of the heart which can act with sufficient force to develop the murmur, and came to the conclusion, from physiological, pathological, and clinical evidence, that it must be the left ventricle.

I will not go into any further details of the original paper, but beg to refer those interested in the subject to it.

I have paid a good deal of attention to the same subject since June of 1897, and will now give an outline of the arguments which seem to me to throw fresh light on the subject, and to confirm my adhesion to the "heterodox" theory, that the true crescendo murmur of mitral stenosis is of early systolic rhythm, and is formed *by blood regurgitating through the stiff jaws of the stenosed valve, whilst they are abnormally slowly closing under the rising force of intraventricular pressure, generated by the earliest phase of the ventricle's contraction.*

NEW ARGUMENTS IN SUPPORT OF THE EARLY SYSTOLIC RHYTHM THEORY—EXPERIMENTAL.—1. Although there can be no doubt in my mind that the *method of production* of an imitation crescendo murmur by air is analogous to that which produces a crescendo murmur in the heart, I carried out similar experiments with water, using an old Higginson syringe with the outlet valve cut off, but with 3 or 4 in. of free outlet tube left. I found that by filling the syringe under water in a basin, and by holding the chest piece of a binaural stethoscope with the left hand to the end of the outlet tube, whilst compressing the ball of the syringe with the other hand, a bruit could be heard which was caused by the flow of water out of the orifice of the outlet tube, and that this bruit rose in pitch as the orifice of the tube was gradually obliterated by pressing it against the rim of the stethoscope (Fig. 2). Here then is an identical experiment to the drainage tube, but carried out with water as the bruit-producing medium; and, moreover, I found that all the variations of an imitation crescendo murmur which can be produced by air can equally easily be developed by water. So my theory, that the crescendo murmur of mitral stenosis is developed by blood rushing through the stenosed valve whilst it is closing, which was formed as the result of experiments with air, is confirmed by similar experiments with water.

2. *Rhythm of the murmur.* — Having found that I could imitate a crescendo murmur with water, I carried my experiment a stage further to investigate the rhythm of such a murmur.

My apparatus consisted of two syringes, coupled together by a tube, and represented the left side of the heart (Fig. 3). The auricle syringe (A) is simply a ball syringe with inlet tube and valve, and a short valveless outflow tube, which is connected with the tube running into the other syringe. The tube represents the auriculo-ventricular ring and the second syringe (V) the

ventricle. This latter syringe consists of an old type of Glover's lithotripsy evacuator, part of which is formed by a glass chamber. Into the free end or head of the glass chamber the auriculo-ventricular tube screws, so that about 1 in. projects into the chamber and an equal amount remains outside. To the outer end of the tube the auricle syringe is fixed, and on to the inner end imitation stenosed valves are placed, where their behaviour, under different conditions of water pressure, can be clearly watched.

The imitation stenosed valves are best made by shaping out of a piece of grey rubber sheet, $\frac{1}{24}$ in. thick, a slightly conical tube about $\frac{3}{4}$ in. long, and with a diameter at the apex equal to about that of a black-lead pencil. The rubber is cemented into the proper shape by means of the solution of rubber in puncture-mending outfits for bicycle tyres. Rather stiffer, and not quite so satisfactory, but still very fair imitation valves, may be made with $\frac{1}{2}$ to $\frac{3}{4}$ in. of rubber drainage tubing, about $\frac{1}{4}$ in. in diameter of lumen. Either of these valves is simply slipped on to the inner end of the metal tube, so that about $\frac{1}{4}$ in. of it projects beyond the end of the tube. The metal tube is then screwed home, and the valve is seen in position through the glass chamber, and it will be noticed at once that it tends to prevent the flow of water out of the ventricle syringe, but allows a free flow in the opposite direction. In this apparatus, then, the syringe (A) corresponds with the auricle, (V) with the ventricle, and the imitation valve with the stenosed mitral valve. There is no outlet from the ventricle syringe to compare with the aorta, but I have purposely avoided such, as without it a high pressure can be more readily generated, and the results obtained are the same as if an aorta were present.

FIG. 3.—Diagram of apparatus for investigating the rhythm of crescendo murmurs.

I will not go into any more detail concerning this apparatus, but will mention the results obtained with it. As before, the experiments were carried out under water to keep all air out of

the apparatus. The object of my inquiry was, to see whether I could produce a crescendo murmur by forcing blood from the auricle syringe into the ventricle syringe, or in the opposite direction. To hear the bruits the chest-piece of the binaural stethoscope must be applied to the glass chamber under water, but I may say that the murmurs are not nearly as distinct as in the former experiment, owing to glass being a bad conductor of sound. To cut a long story short, I found that it was quite impossible to produce any bruit of varying pitch, and nothing at all resembling a crescendo murmur, whilst the water was being forced from the auricle into the ventricle syringe through the stenosed valve; but the moment the current of water through the valve was reversed, by compressing the ventricle syringe, very fair imitations of "blubbering" ascending-pitch murmurs were obtained. If the ventricle syringe be compressed gently—these ventricle experiments are best done after disconnecting the syringes—to imitate a feeble contraction of the ventricle of the heart, water will flow out through the valve, which is stiff enough to resist closure by a slight force of water pressure, and if the syringe be compressed more strongly a bruit of regurgitation ("systolic") will be heard. Further, if more force be used, the escaping water under high pressure throws the valve into "blubbering" sonorous vibrations, which can be felt by the compressing hand as well as heard with the stethoscope. The vibrations of the valve producing this sound can also be *seen* through the glass of the apparatus. They vary in character with the thickness and stiffness of the imitation valve, for, the harder and stiffer the latter, the louder and rougher is the murmur. Finally, if still more force is used, the murmur assumes a crescendo character, and is heard to rise in pitch and to terminate abruptly with a thud comparable to that at the completion of a crescendo murmur in mitral stenosis.

With the less perfect valves a smooth uniform "systolic" bruit may run on from the thud which terminates the crescendo murmur, in the same way as described with my air experiments, and is caused by the presence of a small leak in the otherwise closed valve, probably at the edge (Fig. 2, E, F). This experimental method of producing an imitation crescendo murmur also shows the probability of the abrupt first sound of mitral stenosis being caused by the impact of the walls of the closing rigid valve, for a clear note of impact can be heard when the sides of the rubber valve are seen to be forced into collision by compressing

the ventricle syringe. There is no difficulty in understanding how a note of collision should be heard when the sides of the rigid, often cartilaginous, jaws of a stenosed valve meet abruptly under forcible pressure, even if the colliding surfaces should only travel $\frac{1}{6}$ in. in approaching each other.

ORIGIN OF THE FORCE WHICH PRODUCES THE CRESCENDO MURMUR.—In my previous paper I argued that the auricle is not always, if it is ever, strong enough to contract with that vigour which is necessary for the development of the murmur. My own experience is, that the auricle is as often as not abnormally weak in mitral stenosis, and quite incapable of producing a vigorous murmur. As further support of this belief, I may quote the following figures, obtained from the post-mortem room, which show the condition of the wall of the left auricle in cases of mitral stenosis.

Sansom[1] found the auricular wall hypertrophied in thirteen cases and dilated in eighteen cases of mitral stenosis, whilst Samways[2] found hypertrophy recorded, in the *Guy's Hosp. Rep.*, London, in sixty-five, and dilatation in sixty-eight, of a total of 173 similar cases. Sansom[3] says that "the muscular fibres of the wall of the auricle may be attenuated to almost extinction"; and Balfour[4] writes: "It does seem remarkable and even mysterious that the comparatively feeble and brief contraction of the auricle should be associated with a murmur so loud and rough as the presystolic murmur," nevertheless the facts in favour of such being the case seem "incontrovertible." Any theory which explains the production of a crescendo murmur must hold good in whatever condition the left auricle may be, and it must be remembered that this murmur not uncommonly occurs in cases in which the auricle is dilated and abnormally weak. On the other hand, there is no question as to the ability of the left ventricle to produce a vigorous murmur like the crescendo murmur of mitral stenosis.

DURATION OF A CRESCENDO MURMUR.—The time occupied in the production of these murmurs, as explained by my theory, is only a small fraction of a second. Thus, in a heart beating at the

[1] "Diseases of the Heart and Thoracic Aorta," 1892, p. 353.
[2] *Brit. Med. Journ.*, London, 1898, vol. i. p. 365.
[3] *Ibid.*, 1898, vol. i. p. 1644.
[4] "Clinical Lectures on Diseases of the Heart," 1898, p. 118.

rate of seventy-five per minute, each beat occupies ·8 of a second of time.[1] Of this time ventricular systole takes up ·3 and ventricular diastole ·5. Auricular systole occupies ·1 of the time taken up by ventricular diastole. Less than half the duration of ventricular systole fully covers the period which I believe is occupied by the crescendo murmur, *i.e.* $\frac{1}{5}$ or less than $\frac{1}{5}$ of a second (Fig. 1, A, B, C, D). But though of very short duration, crescendo murmurs may vary in length, some being more prolonged, whilst others are very short and abrupt.

In its more marked appearances a crescendo murmur is of a drawn-out emphatic character, and may be imitated by producing the murmur in a leisurely way with the lips, as described in a former paragraph. On the other hand, it is often so short and abrupt, that careful auscultation is necessary before a trained ear is certain of its presence. One feels that there is "something" which gives an impression of impurity to and terminates with the abrupt first sound. This type of the murmur can be imitated by making a crescendo sound as rapidly as possible with the lips. Between these two extreme types are crescendo murmurs of different lengths, which vary with certain conditions under which the murmur is produced. These conditions may be arranged somewhat roughly in the following groups:—

1. *Rigidity of the valve.*—The more rigid and stiff the valve, the longer will closure be delayed, and therefore the longer will a crescendo bruit produced at the valve be. The extreme of this condition is a rigidity which cannot be overcome, with the consequent development of an ordinary systolic murmur of regurgitation.

2. *Rapidity of development of intraventricular blood pressure.*—The earlier the stage of ventricular systole at which an intraventricular blood pressure sufficiently high to close the rigid valve is developed, the shorter will be the crescendo murmur, and *vice versâ*. The development of high intraventricular blood pressure depends on the degree of the pressure within the aorta, and on the force of the heart's contraction. With a high intra-aortic pressure, or with a strong heart beat, the crescendo murmur runs a short, rapid, and vigorous course, but with the opposite to these conditions prevailing the crescendo murmur will be prolonged and less forcible. If with a very low intra-aortic pressure and feeble heart-beat the intracardiac pressure remains too low to close the valve, then the crescendo murmur will be replaced by a

[1] Allbutt, "System of Medicine," vol. v. p. 470.

uniform true systolic bruit. The latter bruit may often be heard whilst the patient is lying on his back in bed, and yet be immediately replaced by a crescendo bruit when the intra-aortic pressure has been raised, and the force of the heart-beat strengthened by the patient sitting up.

3. *The rapidity or slowness of the heart's action—i.e.* the length of the ventricular systole—also has some effect in determining the length of the crescendo murmur. The quicker the contraction of the ventricle, the shorter will be the crescendo murmur under otherwise similar conditions, and *vice versâ*. The transition from the more prolonged to the rapid type of crescendo murmur is at times observed in the same patient. The more prolonged bruit may be heard whilst the patient is lying at rest and the heart beating quietly, but when any increased exertion—as for instance the change to the erect posture—excites more forcible contraction of the heart, the crescendo murmur is heard to quicken in a very marked manner, and it may even vanish completely, the terminal accentuated first sound alone remaining.

In some most favourable cases the change from a systolic murmur and no first sound to a deliberate, and then rapid crescendo murmur, with terminal accentuated sound, and finally to simply an accentuated first heart sound and no murmur, may be observed in the same patient during rest in bed and subsequent increased action of the heart induced by the erect posture or by walking about. Such a series of events is to my mind a further confirmation of the theory, that an ordinary systolic murmur of mitral regurgitation is but an imperfectly developed crescendo murmur, and points most forcibly to the conclusion that the latter bruit is caused by early vigorous ventricular systole and not by auricular contraction.

Nature of Ventricular Contraction.—The nature of ventricular systole favours the theory that the stenosed valve is closed in this phase of the cardiac cycle. Contraction of the whole ventricle wall is not simultaneous, but is in all probability of a peristaltic nature. Teaching, to my mind, is not definite in saying whether the wave begins at the base and extends to the apex, or *vice versâ*. For my own part I think that the contraction ought to begin at the apex, for this is the thinnest and weakest part of the ventricle, being often not more than $\frac{1}{4}$ in. thick (Quain); whereas the rest of the ventricle wall is two or three times as thick. Thus the contraction beginning at the apex

will protect the weakest part of the ventricle and prepare it for the strain which it will be subjected to by the completion of systole. It is by this apical contraction, or at any rate by the earliest portion of the peristaltic wave, in health, I believe, that the already apposed cusps of the mitral valve are competently locked, and the regurgitation of blood into the auricle during the rest of ventricular systole prevented. Foster[1] recognises a phase of ventricular systole, in which pressure is being got up before the semilunar valves are opened. The time so occupied is ·03 of a second in health. In mitral stenosis, however, the peristaltic contraction is, I believe, too feeble at its onset to *close* the stiff immobile jaws of the valve, but sufficiently strong to force blood to regurgitate through them into the auricle, and to give rise to the commencement of a crescendo murmur. The more forcible contraction of the rest of the ventricular wall, following rapidly and with increasing vigour, develops sufficient intra-ventricular blood pressure to close the valve, thus finishing off the crescendo murmur, which is terminated abruptly by the snap of the closing valve.

By this explanation we account for the development of both the ascending pitch and crescendo characteristics of the murmur which we are considering.

Duration of Ventricular Systole in Mitral Stenosis.—In health, as stated before, ventricular systole occupies three-eighths of the whole time of the cardiac cycle, the rest being attributed to ventricular diastole; and in a heart working at the rate of 75 beats per minute, each beat occupies ·8 of a second of time. If the pulse rate increase in health, the additional beats are obtained without appreciable shortening of the cardiac systole, and almost entirely by reduction of the diastole of the heart.[2]

The advocates of the presystolic theory state that the phase of ventricular diastole is abnormally prolonged in mitral stenosis, to the extent, I take it, of the duration of the crescendo murmur, for the early and late diastolic murmurs when present occupy fully the time attributed to the normal phase of ventricular diastole. Now the crescendo murmur lasts for at least ·1 of a second (see Fig. 1); and if this period of time be added to that already occupied by ventricular diastole (·5), we have this latter phase of the cardiac cycle taking up ·6 and ventricular systole ·2 of the total duration

[1] "Text-Book of Physiology," 6th edition, vol. i. p. 264.
[2] Sherrington, *loc. cit.*

(·8) of the whole cycle. This means that the phase of the ventricle's diastole is three times as long as that of its systole. As ventricular systole is the most stable part of the cardiac revolution (Gibson[1]), such a division of the cardiac cycle is very improbable. It is much more likely that the time occupied by the different phases of the heart-beat in mitral stenosis is as indicated in Fig. 1, B, etc., *i.e.*, practically as in the normal state of affairs. Gibson,[2] in his criticism of my view of the origin and rhythm of the crescendo murmur, groups me with other "early systolic" theorists, as believing that the ventricular systole, when it produces a crescendo murmur, begins earlier than is normally the case. But he has misunderstood my view of the case, whatever other writers may think, for I do not consider that there is any alteration in the time of onset, or even in the duration of the ventricular systole, in these cases. There is plenty of time in the three-eighths portion of the whole cardiac cycle occupied by systole of the ventricle, for the development of a crescendo murmur of ascending pitch, whether it occurs alone or is followed by an ordinary systolic murmur of mitral regurgitation.

The possibility of the existence of a transient systolic murmur, terminating with the first sound and due to regurgitation through an incompetent mitral valve during ventricular systole, is acknowledged by Balfour.[3] In the chapter on curable mitral regurgitation the following passages occur:—"The result of even a slight dilatation (from muscle failure) is the establishment of so-called relative insufficiency, not because the auriculo-ventricular opening itself is dilated—that does happen, but at a later period of the affection—nor because the segments of the valve are unable to close the opening, as one alone of these segments is almost sufficient for this purpose; but because, owing to the separation of the cardiac walls by dilatation of the (ventricular) cavity, the insertions of the *chordæ tendineæ* into the papillary muscles are set so wide apart, and so far from the centre of the ventricle, that the trifling pressure of the auricular blood is unable to bring the valve segments into apposition just before the commencement of the ventricular systole. But any interference with the instantaneous closure of the valve, at the moment of ventricular systole, favours regurgitation, and in these cases this is a constant phenomenon, varying in degree at different times. In all these cases there is a more or less impure first sound audible in the mitral area, or a

[1] "Diseases of the Heart and Aorta," 1898, p. 55.
[2] *Ibid.*, p. 156.
[3] *Op. cit.*, p. 168.

transient systolic whiff may terminate in an apparently normal first sound, and in still other cases a murmur is heard throughout the whole of the systole from its commencement to its end. This is quite in accordance with the account just given. In slight dilatation, at the commencement of the ventricular systole, the valve segments are not in apposition, as they ought to be, and there is some regurgitation, but this ceases as the systole progresses and the valve segments are perfectly closed. When the dilatation is greater, the valve segments never come together, and the regurgitation persists throughout the whole of the systole."

Such opinions seem to me to be strong evidence in favour of the early systolic rhythm of the crescendo murmur. For if in such cases of mitral insufficiency, with mobile valve curtains, as Balfour describes, the development of a transient systolic whiff, terminating in a first sound, is possible, why should it not be equally easy for a similar transient systolic whiff to be developed at a rigid, stiff, narrowed mitral orifice, which most certainly requires an abnormally great force of intraventricular pressure to close it? The transient murmur described by Balfour must in my opinion be of rising pitch, from the fact that it is formed at a gradually diminishing area of leakage in a valve. It is thus an imperfectly developed crescendo murmur.

Application of the Early Systolic Rhythm Theory to the Explanation of the various Clinical Appearances of the Crescendo Murmur.—The early systolic rhythm of the crescendo murmur very easily explains, not only the isolated occurrence of the bruit, but also all the combinations which it may form with other murmurs. Thus—

1. *Crescendo murmur alone.*—This is formed by blood regurgitating under pressure through the valve whilst its jaws are being closed by the rising intraventricular pressure developed by ventricular systole. An accentuated first heart sound terminates abruptly this murmur, and is due to the collision of the thick-hard jaws which guard the stenosed orifice (Fig. 1, B, D).

2. *Crescendo murmur continued by ventricular systolic murmur.*—In this case the former bruit is produced in the same way as when it is heard alone, but the coaptation of the jaws is imperfect, and fails to make the valve water- or blood-tight, so a small stream of blood continues to regurgitate through the valve, whilst ventricular systole persists and gives rise to the uniform

systolic murmur. A more or less abrupt first sound divides these two murmurs (Fig. 1, C).

3. *Crescendo murmur and accentuated first sound, alternating with systolic murmur and less marked or no first sound.*—The occurrence of a crescendo murmur and sharp sound (Fig. 1, D) indicates that the intraventricular blood pressure is raised by ventricular systole sufficiently high to produce a transient regurgitant murmur before it closes the stiff valve with a snap, whilst the production of a ventricular systolic murmur, but no sound, means that (Fig. 1, E) the heart-beat is weak; sufficient pressure is not generated to close the valve anything like completely, no crescendo murmur or abrupt first sound is produced, and ordinary ventricular systolic regurgitation goes on, producing the uniform, true mitral systolic murmur. There is no change of rhythm with the alternation of these murmurs, both occurring during ventricular systole.

4. *Diastolic, crescendo, and systolic murmurs forming one long continuous bruit.*—In this case the diastolic murmur is formed by blood being driven through the stenosed orifice, under the propulsive force of the pulmonary blood pressure and of the contracting auricle, aided by the aspirating power of the dilating left ventricle; the crescendo murmur, by blood regurgitating through the *closing* valve during the earlier half of ventricular systole; and the systolic murmur, by blood escaping through a leak in the otherwise *closed* valve, during the latter half of ventricular contraction. The two last murmurs are separated by the abrupt tapping first heart sound, caused by the impact of the jaws of the orifice of the closing valve (Fig. 1, F).

Auricular systole is an event which occurs immediately before, actually excites, and is practically terminated by, the onset of ventricular systole, so there is no difficulty in understanding how a murmur (diastolic) produced by auricular systole can be apparently continuous with a bruit (crescendo), which begins to develop instantaneously with the earliest onset of ventricular contraction.

Some supporters of the presystolic rhythm theory of the crescendo murmur argue, that the fact that a crescendo murmur sometimes is apparently continuous with a mitral diastolic murmur is evidence in favour of the two murmurs being created by the same force acting with different intensities. But to my mind it is much more probable that the crescendo and mitral systolic murmurs, which are also equally often continuous, are both

caused by different degrees of the same force. In the former instance, the most vigorous portion of the compound murmur is its very termination; and, to bring this about, the auricle—often dilated and powerless—must contract most forcibly when the resistance to its action afforded by the distended ventricle is greatest; whereas, in the other compound murmur, the vigorous portion coincides with the early and more forcible half of ventricular systole, and the weak portion of the murmur with the failing half of the ventricle's phase of contraction (Fig. 1, c).

Area of Audibility of the Crescendo Murmur of Mitral Stenosis.—The *crescendo murmur* is, as a rule, only audible over a small area, not greater than that of a crown-piece, immediately about the apex beat. When traced upwards or outwards from this area, the murmur disappears in the majority of cases, and the only trace of it heard is the terminal abrupt first sound of the heart. But cases are by no means uncommon, in which the crescendo murmur is conducted more widely; and I have myself heard it over the sternum and under the angle of the left scapula.

In Gibson's experience, this murmur, though best heard at the apex, may be conducted more widely than the systolic murmur about to be considered when the two occur in the same case.[1]

The *systolic murmur* of regurgitation through a stenosed mitral orifice is audible over an area very similar to the above, but it is supposed to be conducted more often into the axilla and to the back. Still, in these cases, the systolic murmur is more often than not replaced in the axilla and under the angle of the left scapula by the first sound of the heart. Thus, according to Steell[2] in twenty-eight out of forty-six cases (62 per cent.) in which a systolic murmur was present in the mitral area, this murmur was inaudible at the back. In the remaining seventeen (37·8 per cent.) a murmur was audible at the back; but in sixteen of these cases there was a systolic murmur over the tricuspid area, probably due to regurgitation through this latter valve, and it is quite possible that it was this murmur which was conducted to the back. In eight cases in which a systolic murmur was heard in the mitral, and only faintly or not at all in the tricuspid, area, the murmur was inaudible posteriorly.

Gibson says that this systolic murmur is of maximum intensity at or close to the apex beat, but is conducted in every direction,

[1] *Op. cit.*, p. 168. [2] *Med. Chron.*, Manchester, September 1895, p. 426.

though more commonly to the axilla and the angle of the left scapula.[1]

The *mitral diastolic murmur* is generally audible at the apex. In the minority of cases its area of audibility is limited to the apex, but more usually the murmur can be heard above or internal to the apex beat, as well as over this latter area. Some of the faintest of these mitral diastolic murmurs are heard best, not at the apex but at the junction of the fourth left costal cartilage with the sternum, and between this point and the apex beat. It is an almost unknown occurrence for a mitral diastolic murmur to be conducted to the back.

In Balfour's experience,[2] "the position of maximum intensity of this mitral diastolic murmur" (*i.e.* that of mitral stenosis) "varies in each case; it is usually most distinct at the sternal end of the fourth rib on the left side, though sometimes it is heard in the mitral area, and frequently in the pulmonary area. The soft, slightly musical diastolic murmur audible in such cases in the mitral, or more faintly in the pulmonary area, is undoubtedly mitral in character and may disappear as the disease progresses."

Steell[3] says: "It is more common, therefore, to hear a diastolic murmur both at apex and above than at the apex only," in cases in which the mitral valve is stenosed, and the aortic valves healthy, as far as clinical evidence indicates. Thus out of forty cases of mitral stenosis, in which an undoubted mitral diastolic murmur was heard, either at the apex only or at and above the apex, the bruit was audible in eighteen cases in the former and in twenty-two cases in the latter situations.

In another place Steell says: "Now that I have learned to look for a diastolic murmur *above* the apex in every case of mitral stenosis, the frequency of such a murmur seems to be very great indeed."[4]

Gibson[5] says that the diastolic murmur may be heard more widely than an accompanying "presystolic" murmur is, or it may be the reverse.

In discussing the question of the areas of audibility of the murmurs of mitral stenosis, I have quoted in support of my own views the opinions held by eminent observers who differ from my belief that the crescendo murmur is ventricular systolic in rhythm;

[1] *Op. cit.*, p. 169.
[2] *Op. cit.*, p. 129, note.
[3] *Op. cit.*, p. 431.
[4] *Loc. cit.*, p. 431.
[5] *Op. cit.*, p. 168.

so I cannot be accused of having collected statistics to emphasise my own theories.

The foregoing facts show that—

1. The crescendo murmur is most audible at the apex beat, and is rarely conducted to the left margin of the sternum or to the back of the chest.

2. The systolic murmur of mitral stenosis is loudest at the apex beat, and only in the minority of cases is conducted to the back or to the sternum, and even in these cases it is not improbable that the murmur conducted to the back is of tricuspid and not mitral origin.

3. The mitral diastolic murmur is, in the majority of cases, audible near the left margin of the sternum as well as at the apex beat; in some cases it is most distinct at the sternal end of the fourth rib.

These facts, I submit, show that the area of audibility of the crescendo murmur is more nearly allied to that of the mitral systolic than to that of the mitral diastolic murmur. This is contrary to the opinion of the majority of writers.

Limited Conduction of Crescendo and Systolic Murmurs of Mitral Stenosis.—The most probable explanation of the limited conduction of the crescendo and systolic murmurs of mitral stenosis lies in the fact that the streams of blood which cause these murmurs are very limited in volume, for, with few exceptions, it is impossible for any stream of blood of large volume to escape through an incompetent orifice of a stenosed valve, even were the valve to remain practically wide open during the phase of ventricular systole, instead of being more or less competently closed. The orifices of stenosed valves, in their condition of widest patency, are frequently only sufficiently capacious to admit the tip of one's little finger, *i.e.* from $\frac{1}{4}$ in. to $\frac{3}{8}$ in. in widest diameter —they may even be smaller than this; and when closed, the area of regurgitant leakage is often no more than what one's finger-nail can go into. Even where stenosis is not so extreme, the area of regurgitation is very limited compared with that of severe muscle failure incompetence. In support of this theory, that the limited conduction of crescendo and ventricular systolic murmurs of mitral stenosis is due to the very slight volume of the regurgitant stream, may be advanced the evidence afforded by a study of the areas of audibility of systolic murmurs produced by other forms of mitral regurgitation. In incompetence of the mitral valve, resulting from

acute endocarditis, we have a systolic murmur which, when it is first heard, is only audible at the apex beat of the heart. In a similar way murmurs of regurgitation, due to slight incompetence of the mitral valve, resulting from muscle failure of the heart, are heard only at the apex. Both these types of murmur are caused by the regurgitation of but small streams of blood, and it is not until the disease which is rendering the valve incompetent has made more progress, and has given rise to a considerable area of leakage, that the murmur produced by the regurgitation of blood through the incompetent valve is conducted away from the apex, through the axilla to the back of the chest. It is probable, therefore, that the want of conduction of the crescendo and systolic murmurs of mitral stenosis is not due to any want of intensity of sound, but to the smallness of the regurgitant stream which causes them.

The pitch of a murmur seems to have nothing to do with its conductivity, for shrill musical murmurs may be limited to the apex beat, and low-pitched bruits be audible posteriorly.

Gibson says that it is not always the murmur of the loudest intensity which is conducted farthest, and that it is not easy to determine what particular kind of murmur carries best.[1]

It may be argued that the systolic murmur of mitral stenosis *is* frequently conducted to the back; but, as I stated when considering the area of audibility of this murmur, there is no conclusive proof, in many instances, that such is the case, for it is quite probable that the murmur heard at the back in such cases is of tricuspid origin, and produced by regurgitation of blood through the extensive area of leakage which is generally met with in the tricuspid valve in cases in which there is regurgitation through a stenosed mitral valve. Systolic murmurs heard only at the apex beat, and not at the tricuspid area, of the heart in cases of mitral stenosis, are rarely conducted to the back; still I believe that some few cases may be met with in which the area of incompetence of the stenosed orifice is sufficiently great to allow of the regurgitation of a stream of blood of sufficient volume to cause a bruit which can be heard at the back.

I may mention here a point which, though it perhaps has no apparent bearing on determining the area of audibility of the crescendo murmur, is not without interest. It is that the true apex of the heart is not always in contact with the chest wall in severe cases of mitral stenosis. Dilatation of the right ventricle fre-

[1] *Op. cit.*, p. 171.

quently develops in consequence of the obstruction to the flow of blood through the lungs which is afforded by the stenosed mitral valve. This dilatation makes the right ventricle into a kind of water pad which is capable of abnormally large distension, and as a result of this distension the apex of the left ventricle is displaced backwards from its normal juxtaposition with the anterior chest wall. With this condition of affairs present I believe that many crescendo murmurs are not heard in mitral stenosis, which would be were the left ventricle able to conduct them to the chest wall.

Our knowledge of the principles which govern the area of audibility, or the extent of the conduction of cardiac murmurs, is unfortunately still very rudimentary and unsatisfactory; but, for the reasons just given, it seems very probable that the limited area of audibility of the crescendo and systolic murmurs of mitral stenosis is due to the very limited amount of blood which, by its regurgitation, causes these bruits.

But, assuming that I have entirely failed to prove my point, it is a much more difficult task for the advocates of the diastolic rhythm of the crescendo murmur to explain why this bruit is so very rarely heard at the left edge of the sternum, where faint true diastolic bruits are often best heard.

The Murmurs of Aortic Disease.—There is another not uncommon form of heart disease, in which a marked obstruction to the direct flow of blood through the heart exists, and which is comparable to stenosis of the mitral valve. I mean stenosis of the aortic valve; and it is interesting, and at the same time instructive, to compare the auscultatory signs met with in the two conditions.

In aortic as in mitral stenosis, we hear systolic, diastolic, and, according to some observers, "presystolic" or crescendo murmurs.

The ventricular systolic murmur of aortic stenosis being caused by resistance to the normal direct flow of blood through the heart, is analogous to the bruit produced in a similar way at the mitral orifice, *i.e.* to what is commonly called the ventricular diastolic murmur.

The aortic diastolic murmur being one of regurgitation, is, on the other hand, comparable to the ventricular systolic bruit of mitral stenosis, which is also one of regurgitation.

The crescendo or "presystolic" bruit still remains to be considered, and if my contention, that in mitral stenosis this bruit is

regurgitant, be true, we should expect the analogous murmur of aortic stenosis to be regurgitant also; and this is, I believe, admitted to be the case by all those observers who have described it.

We never hear a crescendo murmur at the aortic orifice during the systole of the left ventricle, that is, during the direct flow of blood into the aorta. An aortic systolic bruit, though it may be rough, is always of a decrescendo type, as would naturally be supposed when we remember that the force which produces the murmur, *i.e.* ventricular systole, is of itself failing, and decrescendo in nature.

The very powerful ventricle then never, by its contraction, produces a vigorous crescendo murmur of ascending pitch at the stenosed aortic orifice. We are therefore justified in assuming that the conditions present are not favourable for the development of such a murmur at the stenosed aortic orifice, during the *direct* propulsion of blood through the heart by ventricular systole; and, arguing by analogy, it seems very probable that such a peculiar type of murmur, which requires very special conditions for its development, should not be produced at the stenosed mitral valve by an entirely analogous series of conditions during the direct propulsion of blood through the heart by auricular systole.

The crescendo murmur of aortic stenosis can, I believe, be produced in a similar manner to its mitral analogue, that is, by blood rushing through the diseased valve whilst the latter is closing abnormally slowly. It is not at all improbable that the thickened, atheromatous, stiff valve curtains met with in aortic stenosis should require an abnormal force to bring them together and prevent the regurgitation of blood into the ventricle. Such abnormal force may be developed at a later period of the "systole" of the aortic wall than that at which the normally thin valves close; and whilst the cusps of the valve are being abnormally slowly closed, blood will regurgitate through the gradually diminishing leak in the valve orifice, and create a short murmur of ascending pitch and increasing force, which will terminate with a modified accentuated second sound appearing after the second pulmonary sound has been heard. Such a bruit would terminate at a distinct interval before the onset of ventricular systole; or the stiffened valve may resist closure by the unaided action of the systole of the aorta, and only yield when the negative pressure of the dilating left ventricle is brought to bear on it as well. In this case the bruit would be more prolonged, and its terminal ascend-

ing pitch portion be close to the first sound of the heart denoting the onset of ventricular systole.

On the other hand, with the opening of these stiff valves, a systolic murmur of diminishing pitch (*ee–oo*) will be created by blood rushing through a gradually enlarging orifice, which is opened abnormally slowly by the increasing force of developing ventricular systole. And this murmur, I contend, is comparable in characteristics and in origin with that decrescendo diastolic murmur which is produced at the stenosed mitral orifice in the first half of ventricular diastole by blood rushing through the opening valve.

For the above reasons, then, I believe that the different murmurs of aortic stenosis are produced by forces and processes which are comparable with those concerned in the development of their mitral analogues; and as the aortic bruits have a rhythmic relation to the systole of the ventricle, so have the corresponding mitral murmurs an analogous rhythmic relation to the systole of the auricle; and, such being the case, the crescendo murmurs of mitral as well as of aortic stenosis are regurgitant and not direct.

TACTILE THRILL ACCOMPANYING CRESCENDO MURMURS IN MITRAL STENOSIS.—A "thrill," synchronous in rhythm with diastolic or with crescendo murmurs, is commonly felt in cases of mitral stenosis when the hand is laid over the region of the cardiac impulse. Such a thrill may be one of two types, according to its rhythm.

1. *Diastolic thrill.*—This tactile thrill is always synchronous with the undoubted mitral diastolic murmurs from which it derives its name. It begins after the cardiac impulse has subsided, and, as timed with the stethoscope, after the second sound of the heart, and always dies away before the onset of the next beat of the heart. In character it is somewhat prolonged, not very vigorous, and is composed of fine vibrations. It is of decrescendo force, becoming less and less vigorous towards its termination, which is never abrupt, but of a gradually fading character; but, when it occurs in association with a crescendo thrill, it may not have died away altogether before the onset of the latter, in which case the two form one continuous thrill.

2. The *crescendo thrill* is synchronous with the murmur of the same name, and is termed the *presystolic thrill* by the advocates of the presystolic theory. Like the murmur from which it derives its name, one of its chief characteristics is, that it increases pro-

gressively in vigour, attaining its maximum at the time of its abrupt termination, which is synchronous with the onset of the true cardiac impulse. It is shorter, much coarser, and more vigorous than the diastolic thrill to which it may be joined, although it more often occurs alone. When continuous with the diastolic thrill, the conjoint thrill fills up nearly the whole interval between the second and first sounds of the heart.

Rhythm of the crescendo thrill.—There is no question as to the rhythm of the decrescendo mitral diastolic thrill, for it undoubtedly is developed during the phase of ventricular diastole, but opinions are at variance concerning that of the crescendo or presystolic thrill.

If my theory of the causation of the crescendo murmur of mitral stenosis be true, namely, that it is due to the regurgitation of blood through the closing jaws of the valve at a very early stage of ventricular systole, and before sufficient force has been developed to close the valve and produce the true cardiac impulse, we should expect, in those cases in which a crescendo thrill is palpable, to feel the thrill immediately before, and ending abruptly with, the heave of the apex which denotes the true cardiac impulse, and which is synchronous with the forcible contraction of the left ventricle and with the production of the first sound of the heart.

That such an event does occur, will not be denied even by the most ardent supporters of the "presystolic" theory.

There is frequently, in my experience, to be seen in cases of "crescendo" thrill, a preliminary agitation of the region of the apex beat, immediately before the true cardiac impulse is observed; and this is due, I believe, to the preliminary ventricular contraction which is trying to close the stenosed valve, and which is really an imperfectly developed cardiac impulse. When, however, the mitral valve is forced to close, and the ventricle has thereby obtained the *point d'appui* of resistance which determines its active systole, it contracts vigorously, and the true cardiac impulse is felt.

The above remarks concerning the rhythm of the apex thrill are also applicable to the generally accepted fact, that the crescendo murmur immediately precedes the throb in the carotid artery which is practically synchronous with the cardiac impulse. For as blood is slipping back through the orifice of the stenosed mitral valve whilst the ventricle is endeavouring to close it, the development of intraventricular pressure of sufficient amount to open the aortic valve is delayed, no blood passes into the aorta,

and so no pulse can be felt in the carotid artery. In health, a distinct measurable period of time ($\frac{3}{100}$ second) elapses after the onset of ventricular systole, before blood is forced into the aorta; and it is quite easy to see how the escape of blood through an open valve in mitral stenosis will further delay the development of the aortic and carotid pulse. During this period of delay, in mitral stenosis, the crescendo murmur occurs, preceding the carotid pulse, as it precedes the impulse of the true, vigorous, working ventricular contraction.

The Sphygmograph and Cardiograph in Mitral Stenosis.—The *sphygmograph* affords no evidence of any value in an investigation of the rhythm of the crescendo murmur of mitral stenosis.

The *cardiograph* is of more use, although the tracings taken of the apex beat in health and disease are interpreted differently by different observers. But in most of the cardiograms of cases of mitral stenosis with crescendo murmur, there are to be seen waves indicating more or less marked movement of the apex immediately before the abrupt rise denoting true ventricular systole develops; and these movements of the apex beat could be accounted for by my suggestion that there is a preliminary systolic agitation of the apex of the ventricle during the time of the closing of the stenosed valve, due to the earliest attempt on the part of the ventricle to contract effectually.

CHAPTER III.

CRESCENDO MURMURS IN VALVULAR DILATATION.

CAN CRESCENDO MURMURS DEVELOP WITHOUT AURICULO-VENTRICULAR STENOSIS?—By accepting my theory, that the crescendo murmur of mitral stenosis is formed by blood rushing through an orifice which is gradually diminishing in area, we can understand how an almost identical type of murmur can arise at valves which are not only not stenosed but even of abnormally large orificial area. That a "presystolic" murmur does arise at times in these latter conditions, has been believed for many years by certain observers. The term presystolic is, however, as mentioned previously, used in rather a vague way by some writers, who apply it to any one, or to the combination of all, of the three distinct murmurs which may occupy the interval between the second and first heart sounds in mitral stenosis, and not to the true crescendo murmur only. In a valuable paper, "On Presystolic Apex Murmur without Mitral Stenosis," Phear[1] adopts this vague nomenclature, for, amongst the forty-six cases which he refers to, there are twelve in which there is no mention whatever of a presystolic murmur, diastolic being the term employed. But I believe that it is quite possible for a crescendo murmur of ascending pitch, but without a typical abrupt terminal sound, to occur when there is no valvular stenosis present.

Under my theory the essential conditions for the development of a crescendo murmur are, that the valve at which it is produced must be only *temporarily incompetent*, that the leak in it must become obliterated *during the production* of the murmur, and that the force which develops the murmur must be vigorous. Given these three conditions, it matters not what state the valve orifice may be in. Like the true crescendo murmurs, these new bruits are always regurgitant. I have shown how I think such conditions can occur in stenosis of the aortic, mitral, or tricuspid

[1] *Lancet*, London, 1895, vol. ii. p. 716.

valves, and I will now give my reasons for believing that there may be only a temporary leak when these valves are incompetent from other causes than stenosis.

Incompetence of a valve results from perverted action of the forces which tend to bring about its closure, and from structural changes in the valve appendages. Three muscular forces are concerned in the normal closure of the auriculo-ventricular valves:—

1. *Contraction of the whole ventricle.*—In health the area of the orifices which the mitral and tricuspid valves protect is greatly diminished during ventricular systole, owing to the diminution in area which the bases of the ventricles undergo during systole. According to Ludwig and Hesse, this diminution reduces the areas in question to about half of their diastolic extent.[1]

2. *The circular muscular fibres*, which surround the same orifices, aid by their contraction in the systolic diminution of the area of the valves.

3. *The papillary muscles* attached to the curtains of the valves form the last of the active muscular forces concerned in the development of a competent closure of the auriculo-ventricular valves.

Intact curtains and chordæ tendineæ are essential for the competency of a valve, but, on the other hand, valves may leak without there being any changes in these structures.

Forcible ventricular contraction is not necessary for the closure of these valves in health, but I must point out that, however much a ventricle may be dilated in disease, it is always more powerful than its auricle is, and therefore more able to produce a vigorous murmur.

Should any cause obstruct the normal action of one or more of the above forces during systole of the ventricle, incompetency of the valve will result and a murmur of regurgitation be heard. This incompetency may be permanent, that is, it may last throughout the whole of each systolic beat—an event which most generally happens—or it may be temporary, and only occupy about the first half of ventricular systole.

Permanent incompetency results in the development of an ordinary prolonged systolic murmur of regurgitation.

Temporary incompetency, on the other hand, with a murmur audible only during portion of ventricular systole, may occur in the following conditions:—

1. *Dilatation of the ventricle* from primary muscle failure, or as a

[1] *Brit. Med. Journ.*, London, 1882, vol. ii. p. 821.

consequence of aortic regurgitation or adherent pericardium. This generally leads to an enlargement or dilatation of the mitral valve, making its orifice too extensive for competent closure by the two curtains at the commencement of ventricular systole (Fig. 4, A). In cases in which the dilatation is extreme, the leakage in the valve is never corrected; but in others, in which the disease has not advanced very far, it is quite possible for the area of the valve's orifice to be diminished at an early stage of the ventricle's systole, by the coaptation of the walls of the base of the ventricle and of the margins of the orifice sufficiently to enable the curtains to cut off the leakage and its attendant murmur (Fig. 4, B and C), and make the valve quite competent. It may only require a very

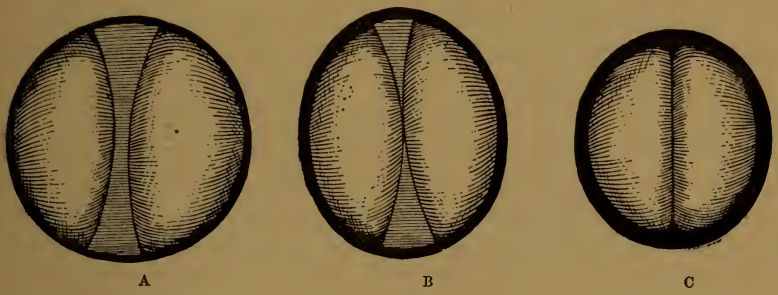

FIG. 4.—Diagrammatic representation of the leakage in a dilated mitral valve, being corrected by the diminution in the area of the valve orifice during ventricular systole.

slight diminution in the area of the valve's orifice to permit of this leak-stopping process.

Balfour, as mentioned previously, when writing on curable mitral regurgitation, states that in his opinion, "in slight dilatation at the commencement of ventricular systole, the valve segments are not in apposition as they ought to be, and there is some regurgitation, but this ceases as the systole progresses and the valve segments are perfectly closed." The result of this is "a more or less impure first sound, or a transient systolic whiff may terminate in an apparently normal first sound." Such a transient systolic murmur would, in my opinion, be of ascending pitch and crescendo force type, from the fact of its being created at a closing orifice and by a force of gathering intensity.

2. The *papillary muscles* also play an important part in the normal closure of the auriculo-ventricular valves. To these muscles the curtains of the valves are attached by means of the

chordæ tendineæ, and it is their function, probably by contractile resistance, to limit the upward movement of the valve curtains during ventricular systole. In health, the bases of the papillary muscles are arranged just far enough apart from each other to allow of the valve curtains moving a sufficient distance upwards to close the valve orifices when contraction of the ventricles begins. When however, as a result of disease, the wall of the ventricles to which the papillary muscles are attached, becomes stretched or dilated, and these muscles are moved abnormally far apart, especially at the onset of ventricular systole, or when the chordæ tendineæ become shrivelled and shortened, the cusps of the valves are dragged down, and have a greatly restricted range of movement, upwards and inwards. In consequence of this, the curtains cannot come as close together at the onset of ventricular systole as they do in health, even if the orifice of the valve be of normal area, but only become competent when the bases of the papillary muscles have to the requisite degree approached each other through the systolic coaptation of the walls of the ventricle to which they are attached. In such a condition of affairs, a leak would occur in the incompetent valve at the beginning of ventricular systole, and disappear at the later phase when the valve is closed up.

Such a temporary crippling of the papillary muscles may result from—(*a*) *Dilatation of the ventricle*, with the bases of the papillary muscles attached abnormally far apart, the chordæ tendineæ and valve curtains remaining normal; (*b*) *shrinking of the chordæ tendineæ* and puckering of the valve curtains, with the cavity of the ventricle remaining practically normal; (*c*) and *adherent pericardium*, which not only acts by inducing ventricular dilatation, but by hindering the systolic diminution of the area of the valve orifices. Foster[1] states that "in health the ventricle away from the apex retires from the chest wall in systole." But, when the pericardium is adherent, such a movement must be impossible, and the diminution in the basal area of the ventricle must be made by restricted cardiac movements, instead of by the free systolic coaptation of the entire basal ring. Here, again, is an opportunity for delay in the competent closure of the auriculo-ventricular valves, when the disease is not too far advanced.

I have not taken into consideration the systolic contraction which the papillary muscles undergo in health (Roy and Adami[2]);

[1] "Text-Book of Physiology," vol. i. p. 238. [2] *Practitioner*, London, 1890.

for, whether this is present or not in the diseased conditions, the results obtained would be the same.

I have now mentioned the conditions besides that of stenosis in which I consider that there may be only a temporary delay in the closure of the auriculo-ventricular valves, and especially of the mitral valve, and I have explained how, in my opinion, a leak which occurs in these valves at the onset of ventricular systole may be corrected completely at a slightly later period of this same phase of the cardiac cycle. So here we have a leak at which a murmur is developed being gradually but rapidly obliterated until it disappears, in much the same way as I maintain occurs in valvular stenosis; and though the area of the leak in the former condition may be greater than in the latter, yet I believe that the murmur produced at the vanishing larger leak will undergo the same changes in pitch as it does in stenosis. What I want to insist on is, that during the obliteration of the leak in dilated valves, a murmur will be developed which will have the character of ascending pitch imparted to it, from the fact of its being caused at an orifice of dimensions gradually lessening to total extinction; and which will be of increasing vigour—*i.e.* crescendo—because it is created during the period of developing vigour of the ventricle's contraction. Such a murmur will have an abrupt termination at the height of its vigour and pitch, but no sharp accentuated terminal sound, as is met with in the crescendo murmur of stenosis, will be heard. If such a leak remains open throughout the whole of ventricular systole, an ordinary systolic murmur of regurgitation results.

The rhythm of the murmur just described is the same as that of the crescendo and ordinary systolic murmurs of mitral stenosis (Fig. 1, D, E).

Crescendo murmur in aortic stenosis.—I have also explained the manner in which I think a crescendo murmur may be produced in aortic stenosis, by blood regurgitating through the abnormally rigid or calcified jaws of the stenosed valve, whilst they are closing, and so receive a character of ascending pitch. A terminal sound, formed by a modified second sound of the heart and not by an accentuated first sound, would be heard in such cases.

Change of Pitch of Non-crescendo Murmurs.—My theory, that a murmur varies in pitch with the variations in size which the orifice at which it is produced undergoes, explains the change from low to high pitch, or *vice versâ*, which is frequently noticed

to take place in ordinary systolic or diastolic murmurs. It is not at all rare to hear a murmur change from low (*oo*) to high (*ee*) pitch during each "beat," and form an imperfectly developed, gradually terminating crescendo murmur. Such a murmur is caused by blood being forced through a valve, in which a leak becomes lessened in extent, but not completely obliterated, during the development of the murmur. It is regurgitant in rhythm, and almost invariably produced at the auriculo-ventricular valve orifice. On the other hand, a murmur may change from high (*ee*) to low (*oo*) pitch by being developed at a gradually enlarging orifice. This type of murmur appears during the direct passage of blood through a stenosed valve, which is forced open more widely by the gathering vigour of the blood pressure behind it.

The changes in pitch here referred to have nothing to do with the variations in force which murmurs may undergo, as a result of changes in vigour of the force developing them.

CHAPTER IV.

CAUSATION OF THE TRUE DIASTOLIC MURMURS OF MITRAL STENOSIS AND OF MITRAL DILATATION.

The Diastolic Murmurs of Mitral Stenosis.—If the view that the crescendo murmur of mitral stenosis is caused during the earliest phase of ventricular systole be accepted, the methods of causation and the positions in the cardiac cycle of the true diastolic murmurs of the same affection must be reconsidered.

There are two of these diastolic murmurs in mitral stenosis, which may be called early and late.

1. *The early mitral diastolic murmur* appears after the second sound of the heart, but in my experience there is always a short sound-free interval between the sound and the murmur (Fig. 1). The murmur is short and decrescendo in vigour and often of descending pitch. It occurs during the earliest phase of active ventricular diastole, and is caused by blood rushing through the stenosed valve into the dilating ventricle, under the forces of the pulmonary blood pressure and of the aspirating ventricle. The diminishing pitch, when it occurs, is caused by the blood rushing through the stenosed valve whilst it is opening up after having been closed by ventricular systole. The method of causation of this murmur, therefore, may be the reverse of that of the crescendo murmur.

The murmur is probably caused by the development of sonorous vibrations in the stiffened rigid valve, and to a less extent by the causation of a fluid vein within the cavity of the left ventricle.

This early diastolic murmur admits of another explanation, especially when the stenosed valve is incompetent and allows of more or less free regurgitation into the auricle. Under such conditions I believe that an abnormal amount of blood collects in the left auricle at the end of the ventricular systole, partly as the result of incomplete emptying of the cavity by auricular contrac-

tion, and partly in consequence of the regurgitation of blood into it during ventricular systole; and into this quantity of blood, more blood, under abnormally high pressure, is poured from the pulmonary veins; and before ventricular relaxation has begun, or at any rate has drawn much blood from the auricle, sonorous fluid veins develop in the auricular cavity and produce a faint, evanescent, early diastolic murmur. This murmur appears very soon after the second sound of the heart is heard, and either disappears quickly as the blood in the auricular cavity is sucked into the ventricle, or else is continued by a murmur developed by the passage of blood through the narrow orifice, in the manner previously described.

2. *The late, or mid-diastolic murmur* in the "presystolic" theory nomenclature, occupies the second portion of diastole of the ventricle, following, and being often apparently continuous with, the early diastolic murmur just referred to. It occurs at the time of, if it is not actually produced partly or wholly by, auricular systole, and could be rightly termed an auricular-systolic murmur, at any rate from a rhythmic point of view. It is caused by the combined forces of the blood pressure in the pulmonary circulation, the aspirating power of the dilating left ventricle, and, in those cases in which the auricle wall is not atrophied, of auricular contraction, driving blood through the stenosed valve whilst its orifice is gaping as widely as possible. The murmur is consequently of uniform pitch, but may be of decrescendo force, owing to the natural failure of the above forces, especially of the contracting auricle, when the ventricle is becoming distended with blood and offers most resistance to their action.

The method of production of this vanishing murmur of decrescendo force, but of uniform pitch, is comparable with the causation of the ordinary prolonged systolic murmur of aortic stenosis, which fades with the termination of ventricular systole.

When the auricular wall is hypertrophied and is acting vigorously, this late diastolic murmur may, with unimpaired intensity, run into and be continued by the true crescendo murmur, which it always precedes in rhythm. It is sometimes continuous with the early diastolic murmur which precedes it, and this compound murmur may be continued uninterruptedly by the crescendo murmur, thus forming one long bruit, extending from the second to the first sounds of the heart (Fig. 1 F, G).

As in the case of the early diastolic murmur, the sound of this

later diastolic bruit probably results from two physical causes—(1) The rush of blood through the narrow orifice, throwing the stiffened curtains into sonorous vibrations; and (2) the development of fluid veins within the ventricular cavity. This last process is to my mind probably of more importance in the causation of the later diastolic than of the early murmur, for we have a much larger mass of blood in the dilating ventricle towards the end of its diastolic phase than at the beginning; and it seems very probable that the small stream of blood which is forced under some pressure by the systole of the auricle into this mass of blood will produce an audible fluid vein. The sound of this vein is readily transmitted to the apex beat of the heart by the medium of the fluid in the distending ventricle. Such a fluid vein murmur would die away with the failure of the force which propels the stream of blood through the stenosed valve.

THE DIASTOLIC MURMURS OF MITRAL DILATATION.—Diastolic murmurs are not uncommonly met with in cases in which the auriculo-ventricular orifices are distinctly dilated, and their valves, in consequence, incompetent. I have already mentioned how a modified crescendo murmur may arise in such conditions, but the origin of a true, non-crescendo, diastolic murmur still remains to be considered. This is, I believe, of fluid vein nature, and arises in consequence of the abnormal anatomical condition of the cavities of the heart met with in cases of ventricular dilatation resulting from various causes, in which the diastolic murmur occurs.

In these cases, the cavities of the auricle and ventricle are much dilated, and their walls are thinned, and of very deficient power. Moreover, their common orifice, which in health is of much smaller area than that of the transverse plane of the distended cavities, becomes much enlarged, rendering its valve quite incompetent, and allowing the cavities of the auricle and ventricle, which it ought to keep separate, to form during the phase of ventricular diastole practically one large, common intracardiac cavity. Owing to the imperfect contraction of the ventricle and the very incomplete expulsion of blood into the aorta and to the free regurgitation into the auricle, which result from ventricular systole, there is at the beginning of ventricular diastole a very large abnormal quantity of blood in the common intracardiac cavity. Into this mass of blood more blood is poured from the pulmonary veins, and a condition of affairs is soon

reached which is favourable to the development of sonorous fluid veins. These are set up by the rush of blood through the orifices of the pulmonary circulation, under often abnormally high pressure, into the blood in the auriculo-ventricular dilatation. The blood in this two-chambered cavity is in an abnormal state of rest, as the walls of the auricle and ventricle, through disease, have lost respectively most of their propulsive and expansile power. It is under these conditions, then, that I believe sonorous fluid veins may arise during the phase of diastole of the ventricle, and give rise to the ordinary decrescendo diastolic murmur met with in cases of cardiac dilatation, resulting from primary muscle failure, adherent pericardium, aortic incompetence, etc. The murmurs so produced may be of varying length.

Difference between aortic and mitral diastolic murmurs.—The diastolic murmurs of mitral and aortic disease, besides being often alike in nature, may both have their point of maximum intensity down the left edge of the sternum, about the level of the fourth rib. This leads to a difficulty in diagnosing the origin of the murmur in the more obscure cases. But there is, I believe, a reliable diagnostic point of difference in the rhythm of the two murmurs in question. I have for some time noted that the mitral diastolic murmur never follows so quickly on the termination of ventricular systole as the aortic regurgitant murmur may do, but that there is always a distinct sound-free interval after the second sound of the heart, or after a systolic murmur, as I have indicated in the diagram of the mitral murmurs (Fig. 1, F, G).

On the other hand, the diastolic murmur of aortic origin, as a rule, follows so quickly after the termination of ventricular systole, that it appears to continue the second sound, when audible, into a murmur, or to be continuous with a systolic murmur, forming a "to-and-fro" or "see-saw" bruit. The diastolic murmur of mitral origin very rarely, if ever, continues the regurgitant systolic murmur in a "see-saw" fashion. But I may point out, as a further analogy between the methods of causation of aortic and mitral stenosis murmurs, that a true auricular-systolic direct, *i.e.* late diastolic, murmur may be continued uninterruptedly by a regurgitant murmur produced at the same orifice.

There seems to be a simple reason for this delay in the appearance of a mitral diastolic murmur. An incompetent aortic valve is only kept from leaking and producing a regurgitant murmur by the blood pressure in the ventricle being greater than that in the aorta, *i.e.* during part of ventricular systole. But the

moment the *vis a tergo* weakens with the completion of ventricular systole, the valves close, the second sound of the heart is heard, and immediately following it, the diastolic murmur caused by the incompetent valve. But whilst the intraventricular pressure falls below that in the aorta practically synchronously with the occurrence of the second sound, there is a pause before the conditions are ripe for the development of the mitral diastolic murmur. If in mitral stenosis, or in cardiac dilatation, this murmur be caused by a fluid vein in the left auricle or common intracardiac cavity, a fraction of a second may elapse before sufficient blood collects to allow of the development of the sonorous vein. If, on the other hand, in mitral stenosis the murmur arises from the blood rushing through the stenosed orifice, a pause always occurs before the ventricle begins its phase of diastolic relaxation, and a further interval may elapse before sufficient force is generated by the aspirating power of the left ventricle, and by the blood pressure in the pulmonary circulation, to drive the blood through the opening, stiffened orifice with sufficient force to generate a murmur. This interval may be more or less prolonged, varying from a very short period of time, as in the early murmurs, to the longer time met with, with later diastolic murmurs.

CHAPTER V.

A CRITICISM OF A CLASSICAL CASE OF TRICUSPID OBSTRUCTION.

HAVING now gone somewhat fully into my theory of the causation and rhythm of the crescendo murmur of mitral stenosis, I will conclude this subject with a reference to Gairdner's case of tricuspid stenosis, which is looked upon by many as the sheet-anchor of the presystolic theory of the murmur in question. In 1861, Gairdner, in a case under his observation, noted a murmur which began immediately after the second sound, continued diminuendo throughout the pause, and then *went on crescendo up to the first sound, at which it stopped abruptly.*[1] Tricuspid obstruction was diagnosed from the presence of the crescendo portion of the murmur, which alone concerns us now, and this opinion was borne out by subsequent post-mortem examination made in 1872, though the true nature of the obstruction, namely, a tumour in the right auricle, was not originally diagnosed. The tumour was 1½ in. in diameter, bullet-shaped and pedunculated in appearance, and of a cartilaginous consistency. It was attached to the posterior wall of the right auricle, whence it hung down so that its lower free extremity was at the tricuspid orifice. Judging from the diagrams accompanying the report of the case, the tumour must have been pushed well down into the tricuspid orifice by the contraction of the auricle, for it was plainly visible from the ventricular side of the valve (Fig. 5). The heart was not appreciably enlarged or altered in any other way.

We are only concerned with the terminal crescendo portion of the compound murmur which was heard in this case, the earlier diminuendo portion being undoubtedly caused by the tumour obstructing the flow of blood through the tricuspid valve into the ventricle. The diminuendo character was imparted to the murmur, in my opinion, by the flow of blood past the tumour

[1] *Edinburgh Hosp. Reports*, vol. i. p. 226.

diminishing in quantity and force, in consequence of the distended ventricle offering more and more resistance to the reception of blood in its cavity. Gairdner considered that the terminal crescendo portion of the long murmur was caused by the contraction of the auricle driving blood past the obstructing tumour with an added vigour to the forces of pulmonary blood pressure and ventricular aspiration, which created the earlier part of the murmur. In his opinion the tumour could only have been obstructive during ventricular diastole and auricular systole, and "could by no possibility become the physical cause of a murmur of regurgitation."

Gairdner further believes that the murmur occurred "under circumstances which render it impossible to suppose that there should have been any difficulty or delay in the closure of the valve, any more than in a normal heart. And even had there been regurgitation, the obstacle, not being fixed but movable, would not have been in the way any more than the ball valve of a syringe is in the way of the current generated by suction."[1] My object now is to show that these conclusions were incorrect, and that the crescendo portion of the long murmur was ventricular systolic, *i.e.* regurgitant in rhythm. Although I have never seen the specimen in question, the case is reported and illustrated in sufficient detail to allow of a criticism of the statement that it was "impossible to suppose that there should have been any difficulty or delay in the closure of the valve any more than in a normal heart" from the presence of the tumour in the auricle. If the tumour were forced by auricular contraction as far into the tricuspid orifice as is indicated by the illustration (Fig. 5) of its position in the flaccid post-mortem heart in Gairdner's paper, it would in my opinion have, of necessity, formed an obstruction to the normal action of the valve, and have made it absolutely impossible for the curtains of the valve to come into competent apposition whilst such an obstruction persisted. Competent closure of the valve would have been delayed until the tumour were expelled, an event which could only result from the rising force of intraventricular pressure; and as an auricle normally remains contracted for an appreciable fraction of time after ventricular systole begins, the development of sufficient blood pressure to compel the expulsion of the tumour and so allow of the consequent competent closure of the valve curtains would not take place, in the case in question, at as early a phase

[1] *Ibid.*, p. 231.

of ventricular systole as that which determines the normal closure of this valve. With the tumour so obstructing the action of the tricuspid valve curtains, a murmur of regurgitation would result,

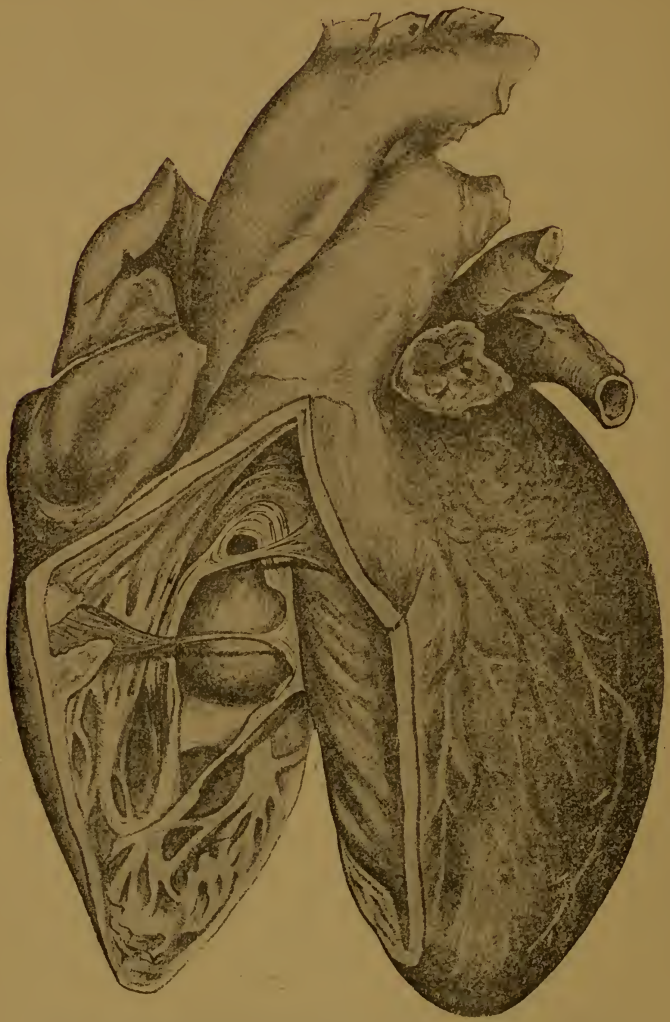

FIG. 5.—After Gairdner, *Edin. Hosp. Rep.*, 1893, vol. i. p. 226.

owing to blood escaping through the gaps in the imperfectly apposed curtains, or between the curtains and the tumour (Fig. 5). As the obstruction was being gradually forced out from between the valve curtains, the extent of the incompetency of the

latter would diminish until complete competency were regained. Through the lessening area of leakage thus formed, blood would escape in the early portion of ventricular systole, and cause a murmur which would rise in pitch, increase in force, and terminate abruptly in the manner previously described in connection with the cases of temporary incompetency of dilated auriculo-ventricular valves.

An imitation of the mechanism by which such a crescendo murmur could be produced by the tumour interfering with the closure of the valve curtains in the above case, can easily be made with the tip of a finger to represent the tumour, and the lips to replace the valve curtains. Blow out the cheeks, keeping the lips tight so as to raise the atmospheric pressure in the mouth, and whilst doing this insert the tip of the finger between the lips and then withdraw it rapidly. During the sudden withdrawal of the finger a small amount of air escapes through the lips and gives rise to a short bruit which is of ascending-pitch type, owing to its being created at an aperture of progressively and rapidly diminishing area. It also terminates abruptly with the final closure of the lips, which prevents any further escape of air.

If this experiment be repeated without applying the lips so closely to the finger and in such a way that the finger, when being withdrawn, is practically blown out of the mouth, the resulting bruit is even more markedly of ascending-pitch character than when it is produced as in the former experiment. I have no doubt whatever that a similar result would be obtained with water as the bruit-producing medium, but owing to practical difficulties I have been unable to devise an apparatus for proof of this.

The comparison of the tumour in the tricuspid valve to a ball valve of a syringe is not a happy simile. I cannot see any resemblance between the act of expulsion, by intraventricular blood pressure, of the tumour from the tricuspid orifice into which it is forced, and where it is held by auricular contraction, and the normal unrestricted movement of an ordinary ball valve resulting from the action of the current which opens it.

For reasons mentioned above, there seems to be no doubt that the tumour acted as a foreign body in the tricuspid valve, just in the same way as would a finger inserted into the valve. Unimpeded action of the three curtains of the valve at the very onset of ventricular systole is essential for the competent closure of the valve, and this would be impossible with the foreign body in the

valve orifice. The auricle exerts no suction action on the contents of the ventricle at the onset, if it does at any portion, of the systolic phase of the latter's action, and the expulsion of the obstructing tumour which prevents the valve from closing would rest with the ventricle. So I fail to see any resemblance between the obstructing tumour in the tricuspid valve orifice and the ball of a ball-valve syringe. The one is an obstruction to the normal action of a "flap" valve, whilst the other is an essential component of the valve to which it gives a name.

I have thus briefly shown what are to me the weak points in Gairdner's deductions from the facts observed in his valuable case, and have offered an alternative explanation of the causation of the crescendo portion of the murmur met with in it, which is in harmony with the theory which I maintain explains the causation of the crescendo murmur of mitral stenosis, and which determines its rhythm in the earliest phase of ventricular systole.

Even assuming Gairdner was right & that the crescendo murmur of his case was A·S in rhythm this helps my theory & very usefully, for the humour was forced further & further into the valve orifice whilst the "diastolic" murmur was being produced as the passage way for the blood became less & less so did the pitch get higher & higher thus shews that my theory of rising pitch at a closing orifice is applicable to be met in cardiac conditions

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